

CASE REPORT

Anesthesia for a long-term anorexic patient with end-stage liver cirrhosis : A Case Report

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Abstract : Recent advancements in intensive care have increased the number of severe anorexia nervosa patients presenting for surgery. We provided anesthesia to a patient who had a 22-year history of anorexia with life-threatening cirrhosis. Although surgery should be avoided in patients with end-stage cirrhosis, she was in the best preoperative optimized condition compared to her condition over the past few years. Potential complications are heart failure easily caused by deterioration of cirrhosis, lethal arrhythmias related to electrolyte disturbances and increased myocardial sensitivity to drugs, and refeeding syndrome in the postoperative period. The several rare events that we experienced are worth reporting. *J. Med. Invest.* 66:337-339, August, 2019

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INTRODUCTION

With an increase in the prevalence of anorexia nervosa (AN), severe AN patients have been presenting for surgery since the 2000s. (1) Patients with a long history of severe AN show several complications, such as acute liver injury, cardiovascular complications, pancytopenia, etc. However, only a few cases of liver cirrhosis have been reported. We experienced a patient with a long history of AN and life-threatening cirrhosis who required anesthesia. We share here our experience with the preoperative evaluation, perioperative events and management of this patient. The patient and her husband have provided “written consent”

CASE REPORT

A 43-year-old female with a 22-year anorectic history was scheduled for endoscopic submucosal dissection for esophageal cancer. Her body mass index was 15.5 kg/m² (height 148 cm ; weight 34 kg). Among her physical symptoms related to the long history of anorexia, cardiovascular complications and Child C cirrhosis appeared to be the biggest problems from the point of view of anesthesia and the endoscopic procedure.

Her preoperative data related to the cardiovascular complications were as follows. Her blood pressure was 81/59 mmHg and heart rate was 99 beats per minute, indicating a class II shock index of 99/81 = 1.2. Preoperative echocardiography revealed a shrunken heart, redundant mitral valve and pericardial effusion. She had a history of high output heart failure with pulmonary hypertension since one year.

Her Child-Turcotte-Pugh (CTP) score was 12 (class C) and the model of end-stage liver disease (MELD) score was 15. According to standard guidelines, patients with MELD scores

above 15 should be considered for transplant. According to the Mayo Natural History Model for Primary Biliary Cirrhosis, the estimated 1-year and 5-year survival probabilities without treatment in such patients are 58% and 1%, respectively. Due to the cirrhosis, her prothrombin time-international normalized ratio was prolonged to 1.5 and blood platelet count was decreased to $6.4 \times 10^4/\mu\text{L}$. Thoracic and abdominal computed tomography revealed ascites and pericardial effusion. Although she had a repetitive history of hepatic encephalopathy, she had not had an episode of encephalopathy since she got married the previous year, and her preoperative ammonia level was normalized at 41 $\mu\text{g/dL}$ (12–66). Other laboratory data related to liver function were aspartate aminotransferase (AST) level of 43 IU/L, alanine aminotransferase (ALT) of 28 IU/L, γ -glutamyltransferase of 35 IU/L, albumin of 2.6 g/dL and total bilirubin of 3.0 mg/dL.

Additional significant preoperative data were hemoglobin 11.2 g/dL, creatinine 1.44 mg/dL, potassium 3.4 mEq/L, phosphate 2.8 mg/dL (<2.3) and magnesium 1.0 mg/dL (1.9–2.5). Rapid turnover protein levels were prealbumin 7.3 mg/dL (22.0–34.0), retinol binding protein 1.2 mg/dL (1.9–4.6) and transferrin 75 mg/dL (190–320). She had repeated histories of electrolyte disturbances, including potassium levels of 2.0 and 1.6 mEq/L, respectively, at the ages of 41 and 42 years. Fortunately, her electrolytes, except magnesium, had normalized over the year after her marriage.

Before induction of anesthesia, 100 μg fentanyl was injected. For induction of anesthesia, awake intubation was planned and administration of anesthetic agents was minimized, including minimum propofol usage and avoidance of muscle relaxants, in view of the loss of muscle mass caused by longstanding malnutrition and abnormal serum magnesium levels. Minimizing the propofol dose also helped obviation of hemodynamic collapse. We used electrocardiogram, non-invasive blood pressure, pulse oximeter and arterial pressure-based cardiac output monitoring system, FloTrac. Before anesthesia induction, Cardiac Index (CI) and Stroke Volume Variation (SVV) were 5.5 L/min/m² and 3%, respectively. No hemodynamic change was observed after awake intubation, but hypotension (BP : 60/35 mmHg) was triggered following injection of 20 mg propofol, with a decrease in CI to 3.0 L/min/m² and elevation of SVV to 30%. Phenylephrine was given to increase peripheral vascular resistance and restore

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hemodynamic stability. Anesthesia was maintained with 0.6–1.2% sevoflurane without a muscle relaxant. Intraoperatively, 25 and 100 µg fentanyl was injected twice when the patient moved or blood pressure was elevated, although with resultant hypotension each time. However, no major complications occurred. After the procedure, the patient was successfully extubated because spontaneous breathing had been maintained during the operation.

Postoperatively, based on the National Institute for Health and Care Excellence (NICE) guidelines for monitoring of refeeding syndrome, serum phosphate levels were measured daily from postoperative days (POD) 1 to 4, which indicated serum phosphate levels of 3.3, 2.2, 2.5 and 3.1 mg/dL, respectively. On POD 2, although phosphate levels decreased to below the lower limit of normal, she did not develop refeeding syndrome. The score gradually recovered over the following days and the patient was discharged on POD 4.

DISCUSSION

We describe a patient with a long history of AN accompanied by end-stage cirrhosis. (2) The patient had a high risk of mortality in the perioperative period, due to potential complications related to the cardiovascular system, hepatic failure and postoperative refeeding.

The expected cardiovascular complications were heart failure and lethal arrhythmias. Patients with severe AN have a low cardiac output and increased peripheral vascular resistance. (3) In contrast, patients with cirrhosis have a hyperkinetic and hyperdynamic circulation with tachycardia, low systemic blood pressure and splanchnic vasodilatation. (4) Our patient had developed high output heart failure the previous year as a result of the excessive vasodilatation and increased cardiac output, which caused imbalance between oxygen demand and supply. Exacerbation of cirrhosis and AN-related vitamin B1 deficiency seemed to cause high output heart failure. (5) We were concerned about our patient developing heart failure in the perioperative period, because anesthetic agents tend to decrease cardiac output and dilate blood vessels. In fact, our patient showed dramatic reduction in CI and had increased SVV on induction of anesthesia. Appropriate precautions and prompt correction of hemodynamic changes were important in preventing perioperative heart failure.

Another probable cause of heart failure in such patients is altered transmitral flow, such as with mitral valve prolapse, mainly because of leaflet-to-left ventricular size mismatch. (6) In patients with a long history of AN, the heart shows structural atrophy that may be related to longstanding hypovolemia. Although our patient did not exhibit mitral prolapse, a redundant mitral valve was observed on echocardiography. We consider it is the most important not to change fluid condition among preoperative state, intraoperative state and postoperative. Vasodilation induced with anesthetic agent were managed only by vasopressor and fluid infusion were avoided.

In severe AN patients, lethal arrhythmias are easily induced by the administration of neostigmine, edrophonium or catecholamines, because of increased cardiac sensitivity. (1) Moreover, our patient was in danger of life-threatening arrhythmias, such as torsades de pointes or ventricular fibrillation, due to her past history of extreme hypokalemia of 1.6 mEq/L. Light anesthesia and avoidance of catecholamines, with judicious use of phenylephrine as needed, succeeded in preventing life-threatening arrhythmia during the endoscopic procedure. (4) The muscle relaxants, vecuronium and rocuronium, were avoided because they are metabolized exclusively by the liver and because it would

have been difficult to administer neostigmine as the neuromuscular antagonist. (7,8) As mentioned above, lethal arrhythmias are easily induced by the administration of neostigmine. We did not want to use rocuronium and sugammadex because there are only few reports of the use of these drugs in severe AN patients. (1) In the immediate postoperative period, we strictly monitored and maintained serum potassium levels in the normal range.

Ninety-day postoperative mortality in patients with CTP class C and MELD score 15–19 are reportedly 54.5% and 14.3%, respectively. (9) Surgery should be avoided if possible in patients with cirrhosis who are child class C or have a MELD score of more than 15. (4) Complications secondary to acute liver failure often result in postoperative death. Hence, preoperative optimizations are recommended, including control of ascites, correction of electrolyte imbalance, improving renal dysfunction, cardiorespiratory assessment, and correction of coagulation. (10) Our patient was in better condition at the time of surgery compared to the previous few years, with recovery from encephalopathy, normalization of electrolytes, decrease of pericardial effusion and improved dietary intake. Serous effusion caused by hepatic hypoproteinemia and longstanding anorexia had also decreased. (6)

Yet, the patient was considered high risk in the postoperative period, because of the probability of developing refeeding syndrome because of longstanding malnutrition. Refeeding syndrome is induced by reintroduction of nutrition to a fasting person. Rapid increase of insulin stimulates the movement of extracellular phosphate, potassium and magnesium into the intracellular compartment, which results in rapid decrease in the extracellular concentration of electrolytes. Hypophosphataemia induces lack of adenosine triphosphate and 2,3-diphosphoglycerate, with a consequent deficiency of energy itself. Any patient with negligible food intake for more than five days is at risk of developing refeeding problems. Our patient developed mild hypophosphataemia (0.6–0.85 mmol/L) on postoperative day 2, after 3.5 days of fasting. According to NICE guidelines, the patient was given phosphate 0.3–0.6 mmol/kg/day orally to prevent refeeding syndrome. (11)

In conclusion, anesthesia and surgery in anorexic patients can precipitate severe complications. Hence, we performed the less invasive treatment of endoscopic submucosal dissection for our patient's esophageal cancer. The patient herself wished to undergo the endoscopic procedure. Our patient was successfully managed with preoperative optimization, appropriate intraoperative precautions and postoperative monitoring.

DECLARATION OF INTERESTS

None of the authors have any conflicts of interest.

DETAILS OF AUTHORS' CONTRIBUTIONS

K.H. : Performed perioperative management and wrote the paper ; M.O. : Performed anesthesia with K.H. ; Y.Y. : Gave advice for writing the paper.

CONSENT FOR REPORTING THIS CASE

Permission was obtained from the patient for submission of her case report for potential publication.

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